INFECTIOUS ANEMIA

(SWAMP FEVER) OF HORSES, MULES, AND DONKEYS

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INFECTIOUS ANEMIA (SWAMP FEVER) OF HORSES, MULES, AND DONKEYS

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Infectious anemia, or swamp fever, also known in some sections of the United States as malarial fever, slow fever, and mountain fever, is one of the most serious maladies of horses, mules, and donkeys. It may be acute or chronic, and is characterized principally by intermittent fever, marked depression, progressive weakness, loss of weight, and edema. On account of its widespread distribution, its insidious nature, and its difficulty of diagnosis, it is of grave concern to owners of horses and mules in all parts of the world. It is of considerable economic importance due to the great losses it causes in horse and mule power.

Distribution and Prevalence

The disease was first reported from Europe in 1843 and now has a worldwide distribution. In addition to Canada and the United States, the disease exists in certain well-defined areas in Germany, Switzerland, Sweden, Norway, Finland, Yugoslavia, Hungary, Russia, Japan, and sections of northern Africa.

In recent years outbreaks have been reported from Uruguay and Venezuela, South America.

In North America it was first recognized as a specific disease of horses in Canada, about 1880. The disease has existed for at least 60 years in the United States. Suspected outbreaks of the disease have been reported from 42 of the 48 States.

In the United States outbreaks of the disease are chiefly confined to small areas and show little tendency to spread. For example, small local outbreaks have been reported from time to time in Idaho, Oregon, Nevada, Montana, Mississippi, Wyoming, Louisiana, Texas, and other States.

Generally speaking, infectious anemia is most prevalent in poorly drained, low-lying sections, but it has been found in wooded sections and marshy pastures at high altitudes. It also appears to be more prevalent when biting insects are most numerous, and during wet years more than dry ones. The active form of the disease makes its appearance in May or June, reaches its height in midsummer, and usually declines late in the fall. Chronic cases may be seen in the winter, and it is possible to produce the disease experimentally at any time.

A number of outbreaks of a severe nature have occurred at establishments where large numbers of horses are assembled and maintained, such as army posts, breeding farms, dude ranches, and biological institutions. During the summer of 1947 such an outbreak in Thoroughbred race horses in New England was promptly controlled through destruction of affected animals and application of strict sanitary

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measures. Outbreaks in 1965 attracted national attention. They occurred among horses at race tracks in California, Illinois, New York, Florida, and Maryland.

A chronic form of the disease which was formerly prevalent among mules on the large cotton plantations in the Mississippi Delta is no longer a serious problem since most mules have been replaced by tractors.

Causative Agent

Infectious anemia is caused by a virus which is in the blood, body tissue, and body secretions of affected animals. Although the infectious nature of the disease was known as early as 1859, the causative agent was not definitely established until 1904.

Under natural conditions the virus appears to affect only equines. It may persist in the host for years. It is apparently present in the blood and body tissues of affected animals at all times and may be eliminated with some of the secretions or excretions, such as the milk, spermatic fluid, saliva, eye and nasal secretions, urine, and feces. The exact nature of the virus still remains a matter of discussion. It has not been cultivated outside the animal body and is unable consistently to reproduce itself in animals other than equines.

Debilitating influences that lower the resistance of an animal, such as overexertion, extreme heat, high humidity, faulty nutrition, improper care and handling, bad sanitation, impure water, and a heavy infestation of intestinal parasites, are not only predisposing factors but also have a marked influence on the progress of the disease in infected animals.

The virus shows considerable resistance against disinfectants, heating, freezing, and drying. Definite findings by Department of Agriculture investigators concerning the action of heat and chemicals on the virus have been put into practical use in formulating requirements for the treatment of antiserums prepared from horses, thus safeguarding against dissemination of swamp fever through the use of such biological products. Biological supply houses operating under Government license are now required to heat all antiserums prepared from horses at 58° to 59° C. (136.4° to 138.2° F.) for an hour, which destroys any infectious anemia virus they may contain.

Animals Susceptible

While most investigators agree that infectious anemia under natural conditions is a specific disease of equines, some workers have reported that young sheep, goats, pigs, rabbits, and doves can be infected under experimental conditions. Infectious anemia in man has been reported by investigators in Germany and Holland but it is probable that man is not very susceptible to the disease.

In the experiments conducted by research workers in the United States Department of Agriculture, equines only were found to be susceptible. Attempts to infect calves, sheep, swine, dogs, cats, rabbits, guinea pigs, rats, mice, and pigeons were without success. Similar results have been obtained by various investigators. Research workers in the United States Army were likewise unable to transmit the disease to many species of native birds and small wild animals of

North America. Various unsuccesssful attempts have been made to transmit the infection and cultivate the virus in developing chicken embryos.

Dissemination and Transmission

Although experimental evidence indicates that the disease may be transmitted by injection of infectious material, by insect vectors, and by ingestion of contaminated material, the common method of its spread under natural conditions is still not definitely known.

Ordinarily the disease appears to spread slowly, occurring mostly in scattered instances. However, during the transportation of great numbers of horses, outbreaks may occur when infected animals are moved into new territory, and when conditions are favorable for transmission and for the exposure of large numbers of susceptible horses. The introduction of susceptible horses into infected areas may also result in outbreaks.

Studies and observations made of a severe outbreak of the disease among race horses in New England during the summer of 1947 indicated that biting flies may have played a part in the early spread of the disease from carriers to healthy horses and that further dissemination was brought about by the promiscuous use of contaminated hypodermic needles and similar instruments.

Under natural conditions the disease appears to spread more rapidly among animals on pasture than among those kept in stables, especially during the season of the year when biting insects are most numerous.

Experimental evidence indicates that: (1) The disease is readily transmitted by the injection of blood or tissue emulsions from affected animals into susceptible ones; (2) minute doses of the virus are infective for susceptible animals; (3) the body secretions or excretions may contain the virus; (4) infected mares may transmit the disease to their offspring and, therefore, should never be used for breeding; (5) the disease may be transmitted by external parasites, including biting flies, mosquitoes, and biting lice; (6) it may spread slowly by long, continuous, intimate contact; (7) carriers probably constitute one of the most common sources of the virus in nature and are chiefly concerned in the perpetuation of the disease.

Since infected animals carry the virus in their blood and since small amounts of virus when injected into susceptible animals are capable of producing the disease, the danger of transmission through the use of contaminated surgical instruments, hypodermic needles, tattooing needles and similar objects cannot be too strongly emphasized.

Forms of the Disease, Symptoms, and Termination

The clinical symptoms are variable and depend to a great extent on the form the disease assumes. Infectious anemia may occur as an acute, rapidly fatal disease or, more commonly, as a chronic affection characterized by intermittent attacks of fever, loss of weight, progressive weakness, marked depression, and dropsical swellings on the lower portions of the body and on the legs. Also, no clinical symptoms may be apparent, though the affected animal carries virulent virus in the blood stream.

In the acute form of the disease the incubation period following injection of infected blood beneath the skin is usually about 12 to 15 days, though it may vary from less than a week to 3 months or longer. The onset is sudden and is manifested by a rise in temperature, which usually goes to about 105° F. but may reach 108°. In the acute form the attacks are usually severe and may be more or less continuous or very frequent. The animal is dejected, the head hangs low, leg weakness is marked, body weight is shifted from one leg to another, and the hind feet are frequently placed well forward under the body. The membranes of the eyes show congestion, followed by brownish to yellowish discoloration. Feed is refused. There may be a slight watery discharge from the eyes and nose and, if the weather is extremely warm, profuse sweating. Frequent urination may also be noted, and in severe cases diarrhea may develop. The attack usually lasts from 3 to 5 days, after which the temperature returns to normal and the animal appears to be well except for a marked loss of weight. Occasionally, however, the initial attack may persist until the animal dies.

Dropsical swellings of the sheath, the legs, the chest, and the under surfaces of the body may occur at any time. Subsequent attacks usually follow, with intervening periods of normality varying from a few days to many weeks or months. When the intervals between the attacks of fever are short, the animal seldom lives more than 15 to 30 days. During the attacks of fever and immediately afterward, there is a reduction in the number of red corpuscles in the blood. During the periods of normality between attacks, the red-corpuscle count is usually normal. (Fig. 1, *A* and *B*.)

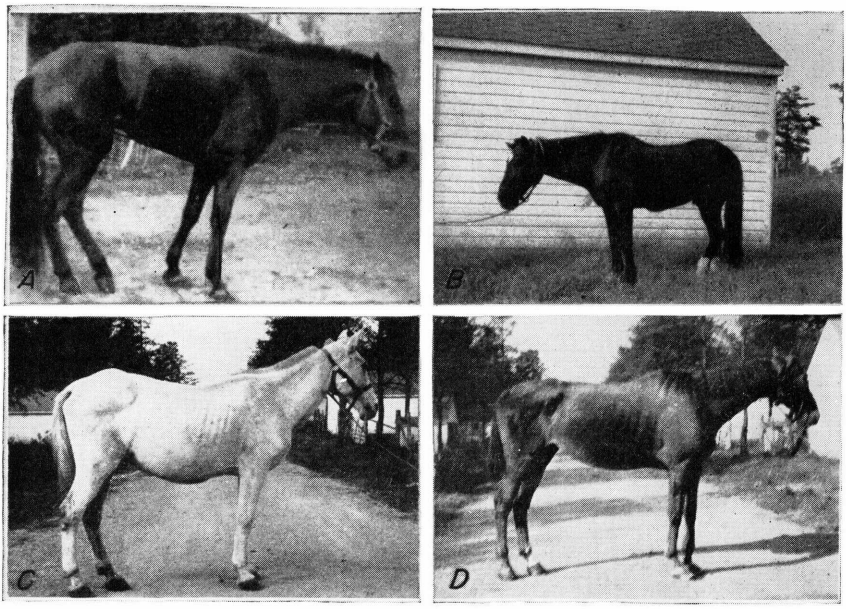

FIGURE 1.—Forms of infectious anemia. *A*—Acute case. Infected by injection of New York virus. *B*—Acute case. Infected by horsefly bites—Wyoming virus. *C*—Subacute case. Field case—Virginia virus. *D*—Chronic case, 9 months after injection of New York virus.

The subacute and chronic forms of the disease differ from the acute in that the attacks are less severe and the intervals between them are longer. The subacute cases may terminate in death during or following one of the attacks, or the reactions may grow less frequent, the animal finally developing into a chronic case or a clinically recovered carrier. In general, the chronic form is manifested by unthriftiness, rough coat, underweight, sluggishness, weakness, dropsical swellings of the lower parts of the body or on the legs, muddy discoloration of the visible mucous membranes, and small hemorrhages on the nictitating membrane (the third eyelid, or haw) and the nasal septum (the partition between the passages of the nose). (Fig. 1, *C* and *D*.)

As the disease progresses, evidence of anemia may develop, the red-corpuscle count may be extremely low, the blood may appear thin and watery, there may be a marked increase in the sedimentation rate of the red blood cells, and in the later stages the visible mucous membranes may become pallid. The pulse may be slow and weak, the heart action may become irregular, and a jugular pulse may be visible. There may be a rapid slowing of the pulse after exercise. Muscular weakness is manifested by a wobbly or rolling, staggering gait or by partial paralysis of the hindquarters. In the chronic form of the disease animals may eat continuously if they have access to feed, but in spite of the excessive consumption of feed, there is a progressive loss of body weight.

Animals affected with this form of the disease can perform some work if handled with care. They are subject, however, to recurring attacks characterized by extreme weakness, knuckling, inability to walk in a straight line, and prominent hemorrhages on the third eyelid. The weakness may become so great that the animal cannot stand without support. With good attention and rest, it usually overcomes these periodic attacks and may go back to routine work. Each attack takes its toll of flesh and strength, however, and repetitions, if frequent enough, will so weaken the animal as to render it useless or finally bring about death by exhaustion.

The inactive or latent form of the disease may follow the initial attack, but it is usually preceded by several attacks of fever. This form is observed in animals that have apparently recovered from the acute, subacute, or chronic types of the disease. Animals affected with the disease in the latent form show no clinical symptoms and are known as clinically recovered carriers. The temperature remains normal, and there is no reduction in the red corpuscles or any sign of disease over a period of years, and yet the infectious agent is always present in the blood stream and all the tissues, and may be eliminated with the body excretions. Such animals obviously are a menace to other horses that may be near them, since they are veritable reservoirs of infection that for the most part go unrecognized and uncontrolled. The inactive form of the disease may, however, become active at any time and present all the characteristics of the acute or subacute form. Unusually hard work or any debilitating influence may reactivate the infection.

The Department of Agriculture had under observation two horses that were good examples of the inactive form of infectious anemia. These animals were first infected in 1935; one harbored the virus in its blood stream for nearly 16 years (fig. 2); the other for 18½ years.

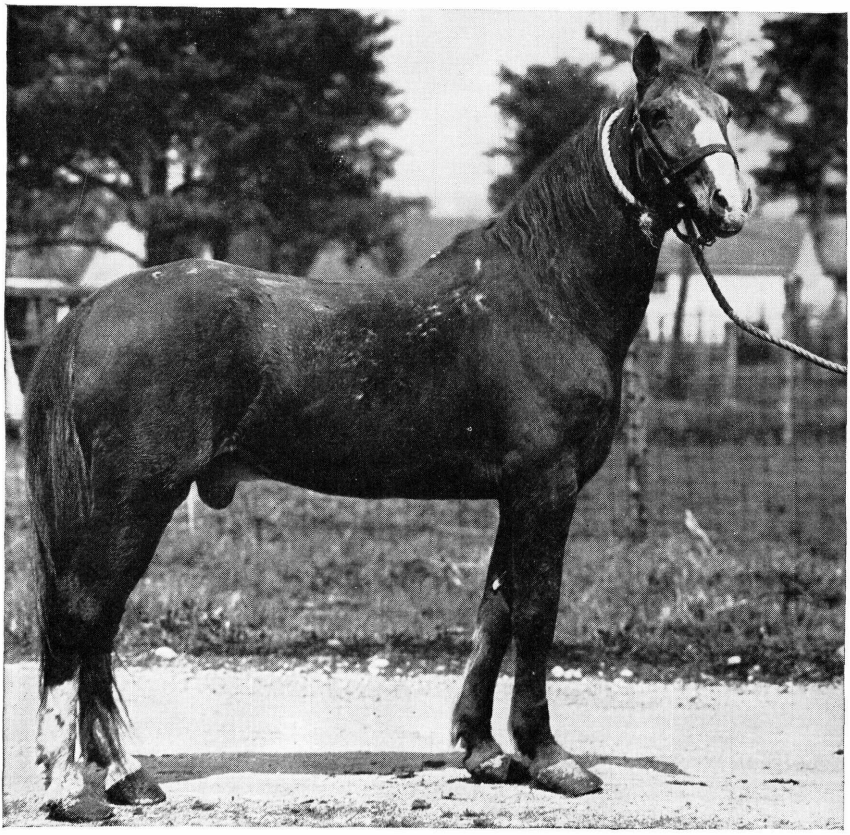

FIGURE 2.—This horse infected in April 1935 carried the virus for nearly 16 years. When this picture was taken he had been a proved virus carrier for more than 11 years and was still in good condition.

Blood drawn from these horses at intervals during these years produced infectious anemia when injected into susceptible horses.

Post Mortem Findings

The most constant lesions of infectious anemia are hemorrhages of varying sizes on the serous and mucous membranes of the body, with enlargement and other changes of the spleen, kidneys, liver, and heart. The hemorrhages are most frequently found on the linings of the body cavities, the heart, the surface of the small and large intestines, the spleen and kidneys (fig. 3).

The spleen for the most part is enlarged. Occasionally it is three times its normal size, and the splenic pulp is soft and blackish red in color. The liver is frequently enlarged to enormous proportions and is hard and easily torn. It may vary from a yellowish-brown, cooked appearance to a reddish-brown color. The kidneys are frequently enlarged, edematous, and lighter in color than normal, and they may show numerous hemorrhages on the surface. The heart may be en-

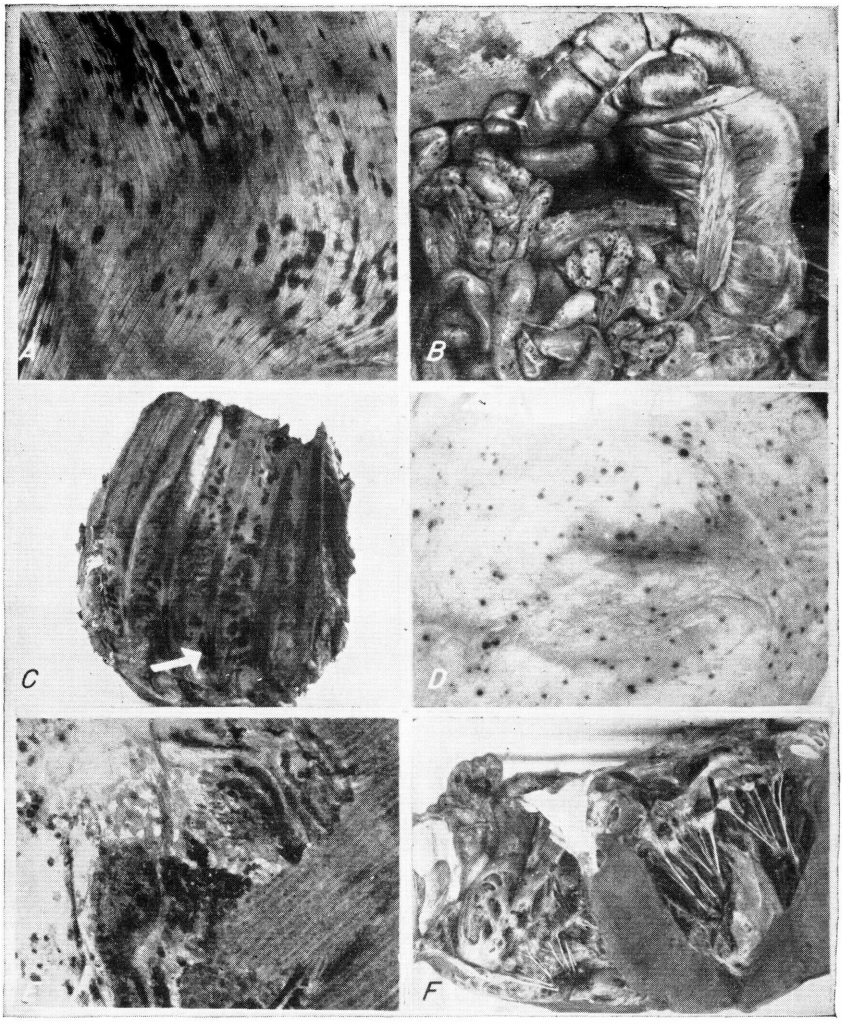

FIGURE 3.—Hemorrhagic lesions in acute infectious anemia. *A*—Abdominal surface of diaphragm. *B*—Serous surface of intestines. *C*—Pleura. *D*—Mucous surface of bladder. *E*—Myocardium (external surface of heart). *F*—Endocardium (lining of inner surface of heart).

larged, flabby, and lighter in color than normal, or it may have a cooked appearance.

The tissue changes in acute and subacute cases are more extensive and more pronounced than those in chronic cases. In chronic cases terminating in death from exhaustion following a protracted illness, emaciation, gelatinous infiltration of fat tissue, and a blanched appearance of mucous membranes are observed. In carriers and chronic cases of a mild type little or no anatomical alteration is observed.

Microscopic examination of the liver tissues of a horse that has died of infectious anemia may show characteristic changes which aid in confirming the diagnosis.

Diagnosis

The greatest obstacles in the diagnosis, study, and control of infectious anemia are the lack of a reliable laboratory test and a suitable laboratory animal for detection of infected animals. Diagnosis of the disease is usually difficult, the only definite means being the horse inoculation test. In active cases a tentative diagnosis based on history, clinical symptoms, blood examinations, and autopsy can be made with a reasonable degree of certainty. For example, a history of rapid loss of flesh, loss of spirit and energy, evidences of muscular weakness with intermittent attacks of fever, congestion of the mucous membranes of the eye, with possibly some degree of jaundice, and dropsical swellings of the lower parts of the body, are strongly suggestive of infectious anemia. The diagnosis will be further strengthened if after the temperature reaction an examination of the blood shows a decrease in the volume of red corpuscles, an increase in the sedimentation rate, and a decrease of hemoglobin. However, in the intervals between the attacks of fever, the blood picture, except in cases accompanied by a progressive anemia, usually returns to normal. The post mortem and histological findings in animals that die furnish additional evidence on which to base a tentative diagnosis.

A tentative diagnosis especially when large numbers of horses are involved in an outbreak should be confirmed by a positive horse-inoculation test, using two or three susceptible test horses and inoculating them subcutaneously with pooled filtered blood serum from the suspected cases.

Infectious anemia in the inactive form is ordinarily not detected, since no clinical symptoms are present to cause suspicion.

In acute cases in the field, the animals may die before the usual train of symptoms develop. The disease in the acute form may be confused with anthrax, influenza, or acute equine encephalomyelitis. In the subacute and chronic forms it may be mistaken for trypanosomiasis (dourine, murrina, and surra) or a heavy infestation with strongyles. The possibility that some of those maladies may be the source of the trouble can be eliminated by laboratory examination.

Since the development of a reliable means of diagnosis is of primary importance from the standpoint of control, a considerable amount of experimental work on diagnostic procedures for the detection of infected animals was carried out by the Department of Agriculture investigators. Results obtained in experimental work with laboratory tests, such as the complement-fixation test, the Fulton mercuric chloride test, Boshian's chlorometric test, the chicken red-cell hemagglutination test of Dreguss, and the blood sedimentation test, all of which have been advocated in the diagnosis of the disease, were frequently indefinite or nonspecific and, therefore, nondependable. Likewise, copious bloodletting, and injections of infectious anemia antigens, or of proteins in large amounts, failed to provoke a specific reaction in known infected animals.

Indefinite results were likewise obtained with the so-called tongue test. This test has been reported as a valuable aid to diagnosis especially in carriers, by investigators in Russia, Sweden, and Switzerland.

Limited studies with negative results have also been carried out by Department investigators with a modified complement-fixation diag-

nostic test recently developed by investigators in Italy, but which is still in the experimental stage.

Treatment and Vaccination

There is no known treatment that will cure the disease. In searching for an effective treatment, many investigators have tried numerous agents such as arsenical preparations, quinine, various dyes, mercurial preparations, and a number of others, but without success. The Department of Agriculture investigators experimented with merthiolate, crystal violet, trypan blue, arsenical preparations, formin, hydrochloric acid, potassium permanganate, fuadin, sulfanilamide, penicillin, and other preparations, using both acute and chronic cases for these tests. None of the preparations, however, exerted any appreciable influence on the course of the disease, nor did any free the infected animals of the virus. In sections where the disease is endemic, or constantly present, practicing veterinarians employ arsenical compounds, principally sodium cacodylate, together with tonics, rest, and abundance of good feed, at the same time eliminating intestinal parasites and other debilitating factors. While such treatment brings about some clinical improvement, it has no lasting value, for the animal remains infected, is subject to febrile attacks, and is a virus carrier. To establish a complete cure, a method of treatment must be found that will not only free the animal of clinical symptoms but completely eliminate the virus from the tissues of the affected animal.

Preventive vaccination has been attempted without success by a number of investigators, including those in the Department of Agriculture. In the investigations conducted by the Department none of the tissue vaccines prepared from whole blood, blood serum, emulsions of spleen tissue, or emulsions of brain tissue in which the virus was destroyed by formalin, crystal violet, phenol, phenyl mercury acetate, and heat had any appreciable immunizing value.

Control

The control of this disease, because of its obscure nature, the difficulty of diagnosis, its resistance to treatment, and its widespread distribution, presents a serious problem. While no systematic program can be undertaken until a definite and practical means of diagnosis of chronic carriers is developed, the following measures constitute the most effective known means of control.

When a definite diagnosis of infectious anemia has been made, it is advisable, if practicable, to kill the animal and dispose of the carcass by cremation or deep burial to prevent further spread of the infection. This method of control has been followed in small isolated outbreaks and in establishments keeping large numbers of horses, and it has been effective. However, this method is impracticable in such areas as the Mississippi Delta, where the disease is widely distributed and exists principally in a mild chronic form.

Animals known to be infected and those suspected should be isolated from healthy animals. Flies and mosquitoes should be controlled. As the disease may exist in the inactive form, with the animals carrying the contagion in the blood stream, the greatest care

should always be taken to prevent transmission of the disease from animal to animal by the use of unsterilized instruments. Clean and sterilize all instruments, such as knives, hypodermic needles, bleeding needles, and tattooing instruments before using them on each animal.

Do not use equipment that may produce skin abrasions or absorb body excretions or secretions, such as bridles, harness, saddles, blankets, brushes, and currycombs, on both infected and healthy horses.

Only horses known to be free from the disease should be used as donors for blood transfusions. All antisera of equine origin intended for treatment of horses should be heated to destroy the virus. Mares or stallions suspected of being affected should not be used for breeding purposes. Horses from areas where the disease exists should be isolated, have their temperatures taken and recorded daily, and be kept under observation for 60 days after being brought on premises where normal horses are kept. In places where large numbers of horses are assembled from various parts of the country, such as race tracks, horse shows, and county fairs, it is advisable that all horses be kept in separate, clean, well-ventilated stalls free from flies, and fed and watered from separate containers. No equipment of any kind should be used interchangeably on the horses.

In endemic areas, known infected animals and healthy animals should not be kept together in small, poorly drained paddocks adjacent to stables and manure dumps. In those areas where there is reason to believe that many animals are affected with the disease, the maintenance of good sanitary conditions, systematic control of parasites, provision for a supply of pure, fresh drinking water and attention to the feed, care, and handling of animals will help to hold the ill effects of the disease to a minimum and retard its spread.

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